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The relationship between systemic immune-inflammation index and length of hospitalization in acute pancreatitis

Systemic immuno-inflammation index in acute pancreatitis

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Abstract
Aim: To determine the relationship of the systemic immune-inflammatory index (SII) in acute pancreatitis (AP) with disease severity and length of stay in hospital.
Material and Methods: This retrospective study was conducted in the Emergency Department over a 3-year period. The basic and laboratory data were examined from the electronic patient records system of 100 patients who were hospitalised because of pancreatitis.
Results: Evaluation was made of 100 patients, comprising 71 females and 29 males, with mild AP determined in 67 and severe AP in 33. The SII and neutrophil-lymphocyte ratio (NLR) values were determined to be significantly higher in the severe AP group (p=0.001, p=0.014, respectively). A statistically significant correlation was determined between the SII and the Glasgow (Imrie) score (r=0.430, p<0.001). According to the Glasgow (Imrie) criteria, the SII was determined to have sensitivity in the differentiation of severe AP. The SII, C-reactive protein level, and Glasgow (Imrie) criteria were determined to be independent predictors of length of stay in hospital.
Discussions: The SII was determined to be effective in predicting disease severity and length of stay in hospital in AP patients.

Keywords
Acute Pancreatitis, Inflammation, Systemic Immune-Inflammation Index, Hospitalization

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Introduction

Acute pancreatitis is a common inflammatory disease of the exocrine pancreas, with symptoms ranging from severe abdominal pain to pancreas necrosis. It can cause permanent organ failure and multiple organ dysfunction and has a mortality rate of 1-5% [1]. At the molecular level, the triggers of acute pancreatitis cause damage to the pancreatic acinar and ductal cells by disrupting the intracellular calcium signal. In acute biliary pancreatitis, high pressure occurs as a result of gallstone obstruction of the ampulla vaterin, and with this high pressure, bile enters the pancreatic canal [2].

Multiple cytokines, primarily tumour necrosis factor-α, and interleukins-1α, 1β, 6, and 18, mediate a strong proinflammatory immune response, exacerbate the initial pancreas damage, and through lymphatic and systemic circulation pathways, inflammatory responses pass to the liver, lungs, heart, kidneys, and gastrointestinal system. This situation progressing in acute pancreatitis causes systemic inflammatory response syndrome [3, 4]. Therefore, studies have been conducted on macrophage-related inflammatory response and neutrophil infiltration associated with acute pancreatitis [5]. The inflammatory response can be determined with the levels of neutrophils, lymphocytes, thrombocytes, and acute phase proteins.

In the determination of the severity of acute pancreatitis, scoring systems are used such as the Acute Physiology and Chronic Health Evaluation II (APACHE-II), the Ranson Criteria or Modified Glasgow Acute Pancreatitis Severity Score (Glasgow Imrie Score) for mortality, and the Atlanta Classification for systemic inflammatory response syndrome. The current guidelines recommend observation of the presence of systemic inflammatory response syndrome or organ failure for a minimum of 48 hours from the time of hospital admission for the prediction of the course of disease severity [6, 7]. Therefore, the scoring systems suitable for use at the time of presentation in the Emergency Department (ED) are limited. Previous studies have determined that the Atlanta and Glasgow (Imrie) scoring systems can be used on presentation at ED [8]. Recently, the systemic immune-inflammation index (SII), which is a combined tool, has been used to obtain prognostic information in patients with various malignant tumours [9, 10]. There are also more recent studies showing that the SII can be used in pancreas cancers and in the early determination of the severity of acute pancreatitis [11, 12].

The aim of this study was to determine the effect of the SII on a change in disease severity according to the Glasgow (Imrie) criteria and the duration of hospitalization in patients diagnosed with acute pancreatitis in the ED.

Material and Methods

Approval for this retrospective study was granted by the Local Ethics Committee. The basic and laboratory data in the electronic patient records system were examined of 100 patients (71 females, 29 males) diagnosed and hospitalised with a diagnosis of acute pancreatitis made in the ED over a 3-year period. The diagnosis of acute pancreatitis was made from the presence of two of three criteria: (1) abdominal pain consistent with pancreatitis, (2) serum amylase or lipase ≥2-fold more than the normal upper limit, (3) findings consistent with pancreatitis on computed tomography (CT) or magnetic resonance imaging (MRI) [7]. The Glasgow (Imrie) criteria were used in the determination of the severity of AP (13]. The Glasgow Imrie score is formed of 8 criteria, each scored with 1 point. A score of ≥3 is evaluated as severe AP [14].

The patients included in the study were aged >18 years and were diagnosed with acute pancreatitis according to the diagnostic criteria. Patients were excluded from the study if they were aged <18 years, if they refused treatment and hospitalization despite the medical recommendation, or if they had a history of chronic hematological disease.

Laboratory measurements:

The neutrophil-lymphocyte ratio (NLR) and SII were calculated from the laboratory results: NLR = neutrophil count /lymphocyte count, and SII = thrombocyte count x NLR [15].

White blood cell (3.7–10.1 10e3/μL), hemoglobin (12–18 g/dL), hematocrit (35%–53.7%), platelet (142–424 103/μl), lymphocyte (1.09-2.99 103/μL), neutrophil (1.63-6.96 103/μL), monocytes (0.24-0.79 103/μL), eosinophil (0.03-0.44 103/μL), mean platelet volume (6.8-10.8 fL), and red blood cell distribution width (11.8-15.8%)counts were determined with the Alinity HQ device (Abbott, USA). The levels of serum glucose (74–106 mg/dL), urea (19–50 mg/dL), creatinine (0.55–1.02 mg/dL), aspartate aminotransferase (13-40 U/L), alanine aminotransferase (7-40 U/L), bilirubin (0.3-1.2 mg/dl), albumin (3.2 - 5 g/dL), calcium (8.7-10.4 mg/dl), lactate dehydrogenase (120-246 U/L), amylase(30-118 U/L), lipase(12-53 U/L), and C-reactive protein (0-0.05) were measured using conventional laboratory methods on Atellica Solution (Siemens Healthineers, Germany). Activated partial thromboplastin time (22-36 sec), prothrombin time (10.5-15.5 sec), and International Normalized Ratio (0.8-1.2 INR) values were determined using the Sysmex CS-2000i device (Siemens Healthineers, Germany).

Statistical analysis

Data were analyzed using SPSS 21.0 (IBM Corporation, Armonk, NY, USA) and University licensed Microsoft Excel packaged software. Numerical data were expressed as median and interquartile range (IQR) values and qualitative data as number (n) and percentage (%). The conformity of continuous variables to normal distribution was determined using the Shapiro–Wilk normality test. In the comparisons of two independent groups, the Mann Whitney U-test was applied and in the comparisons of categorical data, the Chi-square test. Spearman correlation ananlysis was performed to determine correlations. ROC analysis was applied to evaluate the use of SII in the differentiation of severe and non-severe AP. The ROC curve analysis results were presented as % specificity and % sensitivity [area under the ROC curve (AUC), p-value, and 95% confidence interval (CI)]. In the determination of factors affecting the length of stay in hospital, linear logistic regression analysis was used. A value of $p < 0.05$ was considered statistically significant.

Ethical Approval

Ethics Committee approval for the study was obtained.

Results

Evaluation was made of 100 patients, comprising 71 females and 29 males, with mild AP determined in 67 and severe AP in 33. The SII and NLR values were determined to be significantly

higher in the severe AP group (p=0.001, p=0.014, respectively). The basic and biochemical data of patients with non-severe AP (0-2) and severe AP (≥3) according to Glasgow (Imrie) criteria are presented in table 1 and hematology data in table 2.

A statistically significant positive correlation was determined between the SII and the Glasgow (Imrie) score (r=0.430, p<0.001). When a cutoff value of 2178.33 was taken in the ROC analysis, the use of SII in the differentiation of non-severe AP (0-2) and severe AP (≥3) according to the Glasgow (Imrie) criteria, had 72.73% sensitivity and 58.21% specificity (Figure 1).

According to the results of the linear logistic regression analysis, the SII, CRP, and Glasgow (Imrie) criteria were determined to be correlated with the length of stay in hospital (Table 3).

Discussion

The results of this study demonstrated that as the severity of acute pancreatitis increased, so the SII significantly increased, and the use of the SII in the differentiation for severe pancreatitis was determined to have 72.73% sensitivity and 58.21% specificity. A correlation was also determined between the SII and length of stay in hospital.

Although the pathophysiology underlying the effect of locally occurring damage in AP on the systemic inflammatory response is not fully known, neutrophils, monocytes, macrophages, and lymphocytes have been determined to have a very important role in the progression of immune system diseases [16]. Previous studies have based the definition of severe AP on organ failure, and it has been reported that patients with

Table 1. The basic and biochemical data of the patients with non-severe AP (0-2) and severe AP (≥3) according to the Glasgow (Imrie) criteria

Parameters	Non-severe AP	Severe AP	p
N (F/M)	67 (46/21)	33 (25/8)	0.311
Age (years)	49.00 (36.25 - 62.75)	68.00 (48.75 - 81.00)	0.001
Biliary AP n (%)	23 (65.7)	12 (34.3)	0.017
Non-biliary AP n (%)	44 (67.7)	21 (32.3)	<0.001
Length of stay in hospital (days)	3.00 (3.00 - 5.00)	6.00 (3.00 - 7.00)	0.000
Modified Glasgow prognostic score	1.00 (1.00 - 2.00)	4.00 (3.00 - 5.00)	0.000
Glucose	115.00 (103.25 - 138.50)	123.00 (111.00 - 159.00)	0.122
Albumin	3.90 (3.60 - 4.27)	3.70 (3.40 - 3.92)	0.055
Alanine aminotransferase	203.00 (114.00 - 370.00)	257.00 (118.75 - 402.00)	0.395
Amylase	1161.00 (474.25 - 1979.25)	1048.00 (271.25 - 2014.25)	0.647
Aspartate aminotransferase	171.00 (91.00 - 295.75)	174.00 (77.25 - 490.00)	0.528
Bilirubin	1.70 (1.10 - 3.67)	3.30 (1.80 - 4.81)	0.046
Calcium	8.80 (8.42 - 9.40)	8.50 (8.07 - 8.97)	0.045
C-reactive protein	1.30 (0.43 - 5.08)	2.32 (0.58 - 6.57)	0.439
Creatinine	0.70 (0.60 - 0.85)	0.70 (0.70 - 0.99)	0.146
Lactate dehydrogenase	341.00 (270.70 - 433.00)	401.00 (289.75 - 578.25)	0.143
Lipase	985.00 (407.00 - 1786.00)	700.00 (400.00 - 1700.50	0.369
Urea	26.00 (19.26 - 31.57)	37.00 (29.67 - 54.75)	0.000

Table 2. The hematology data of the patients with non-severe AP (0-2) and severe AP (≥3) according to the Glasgow (Imrie) criteria

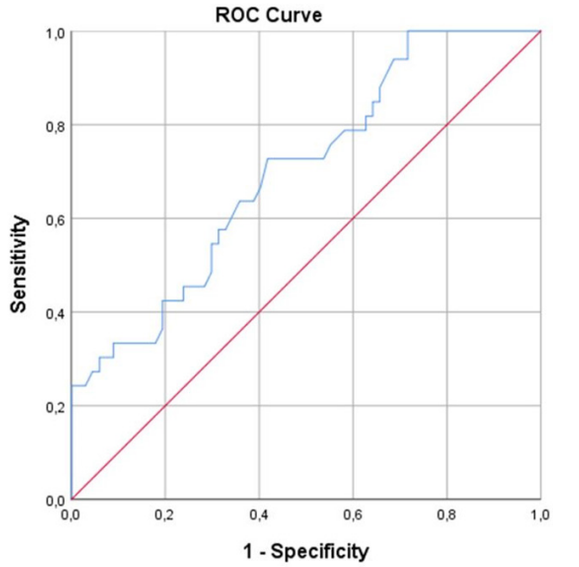
Parameters	Non-severe AP	Severe AP	p
Hemoglobin	13.75 (12.60 - 15.15)	13.30 (11.50 - 15.00)	0.276
Hematocrit	41.71 (37.70 - 46.52)	41.20 (36.05 - 45.62)	0.399
Neutrophil	8.90 (5.91 - 13.07)	11.03 (8.34 - 16.52)	0.048
Lymphocyte	1.20 (0.90 - 1.80)	1.17 (0.80 - 1.53)	0.106
Eosinophil	0.02 (0.01 - 0.07)	0.02 (0.01 - 0.07)	0.686
Monocytes	0.60 (0.320 - 0.86)	0.63 (0.36 - 1.01)	0.673
Mean platelet volume	7.79 (7.07 - 8.87)	7.90 (7.25 - 8.98)	0.505
White blood cell	10.72 (9.00 - 14.86)	13.10 (10.72 - 18.61)	0.051
Platelet	257.00 (198.20 - 320.00)	262.00 (221.70 - 330.00)	0.329
Red blood cell distribution width	12.24 (11.50 - 13.60)	12.50 (11.80 - 15.06)	0.150
Systemic immune-inflammation index	2027.89 (727.13 - 3105.80)	2794.02 (1842.14 - 5231.91)	0.001
Monocyte lymphocyte ratio	0.30 (0.20 - 0.75)	0.52 (0.39 - 0.84)	0.055
Neutrophil lymphocyte ratio	9.05 (4.65 - 11.68)	9.89 (6.75 - 23.69)	0.014
Platelet Mass Index	1940.85 (1727.32 - 2374.57)	1976.00 (1728.61 - 2974.86)	0.319
Prothrombin time	12.60 (12.00 - 13.50)	12.50 (11.70 - 13.62)	0.519
Activated partial thromboplastin time	25.60 (23.90 - 29.47)	24.90 (22.50 - 27.10)	0.099
International Normalized Ratio	1.02 (0.95 - 1.09)	1.02 (0.93 - 1.12)	0.974

organ failure ongoing for more than 48 hours have a greater risk of mortality. Moreover, there is a period of significant inflammatory response (SIRS) before organ failure and this shows that the patient is at very high risk of organ failure [17]. Generally, the majority of patients (80-85%) have a mild form of the disease which is self-limiting and has a mortality rate <1-3%, but approximately 20% of patients have a moderate or severe AP attack, for which the mortality rate increases to 13-35%. Therefore, the early identification of severe AP or predicting that there could be severe AP is of critical importance to further reduce complications and mortality [17].

Table3. Factors affecting the length of stay in hospital

Model	Beta	t	p	95,0% CI	
(Constant)		1.248	0.216	-2.204	9.603
SII	0.962	2.638	0.010	0.000	0.002
CRP	-0.243	-2.403	0.019	-0.119	-0.011
Glasgow score	0.316	3.031	0.003	0.161	0.779
Platelet	-0.765	-1.683	0.096	-0.041	0.003
RDW	0.056	0.542	0.590	-0.141	0.246
NLO	-0.255	-0.920	0.360	-0.245	0.090
WBC	0.020	0.010	0.992	-1.457	1.472
LYM	0.281	0.914	0.364	-0.987	2.659
NEU	-0.189	-0.101	0.920	-1.577	1.425
MONO	-0.054	-0.186	0.853	-3.078	2.553
EOS	-0.093	-0.756	0.452	-9.227	4.150
MPV	-0.217	-0.873	0.386	-0.841	0.329
Age	0.100	1.134	0.260	-0.008	0.030
Amylase	0.035	0.379	0.706	0.000	0.001
Lipase	-0.156	-1.485	0.142	-0.001	0.000

*Standardized Coefficients Beta, SII: Systemic immune-inflammation index, CRP: C-reactive protein, RDW: Red blood cell distribution width, NLR: Neutrophil lymphocyte ratio, WBC: White blood cell, LYM: Lymphocyte, NEU: Neutrophil, MONO: Monocytes, EOS: Eosinophil, MPV: Mean platelet volume



	Cut-off	AUC	95% CI	p	Sensitivity	Specificity	PPV	NPV
SII	>2178,33	0,699	0,600 -0,787	0,0003	72,73	58,21	46,2	81,2

Figure 1. The use of SII in the differentiation of severe and mild pancreatitis

Although many biomarkers have been examined as early predictors of AP severity, there is no practical, consistent, or accurate laboratory test to predict disease severity in patients with AP [17, 18]. The need for at least 48 hours of observation to determine organ failure and systemic inflammatory response limits the use of these scoring systems in the ED. Therefore, studies of biochemical markers to determine AP severity are ongoing. The recently defined SII, which is a combined tool to provide prognostic information in patients with different malignant tumours, has been used in studies [9, 10]. The SII is a simple, low-cost, objective index, which better reflects the balance between the inflammatory and immune responses of the host than all the other systemic inflammation scores. Wu J. et al. reported that the SII increased in ankylosing spondylitis, which is an inflammatory disease of unknown etiology, and as it increased more in periods of disease activity than in periods of remission, it could be used as a biomarker in the observation of disease activity [19]. In another study by Yorulmaz et al., it was reported that SII could be an independent prognostic marker for patients with psoriasis and psoriatic arthritis [20]. It has also been reported that in addition to SII being affected in chronic inflammatory disease and cancer, the SII is affected in acute inflammation conditions [21].

There are several studies in literature that have investigated hematological parameters in AP, which is an inflammatory process. In recent years, the relationship has been investigated of new inflammatory markers to predict the severity of pancreatitis such as the NLR and platelet-lymphocyte ratio (PLR) with prognosis and severity in AP (22). NLR was found to have 58.33% sensitivity and 73.73% specificity in the determination of AP and organ failure in a study by Liu G. et al., and was therefore said to be a marker with prognostic value in AP [22]. Similarly, Zhou et al. reported that NLR, PLR, red blood cell distribution width (RDW) and blood urea nitrogen (BUN) values were good markers for the prediction of AP severity and prognosis [23].

In severe pancreatitis, IL-1, IL-6, IL-8, TNFα, thrombocyte activating factor (PAF), and various cytokines in the circulation can activate chemotaxis, neutophils, monocytes, lymphocytes and thrombocytes [24]. The SII, which is obtained by multiplying the NLR and PLT values, and has been determined to significantly increase in several cancers, has been reported to be important in pancreas cancers.

Bittoni et al. reported that increased SII was a negative prognostic factor independently of others for both overall survival and progression-free survival in advanced pancreatic ductal adenocarcinoma patients treated with first-stage chemotherapy [25]. Liu X et al. reported that SII was more sensitive and specific than NLR and PLR in predicting acute pancreatitis severity and in differentiating AP patients into two groups of moderate and severe according to the Atlanta classification of acute pancreatitis. In that study, SII was determined to have 92.9% sensitivity and 87.7% specificity in the differentiation of moderate and severe AP [12]. In the current study, when the AP patients were separated into two groups of severe (≥3 points) and non-severe according to the Glasgow (Imrie) criteria, SII was found to have 72.73% sensitivity and 58.21% specificity in this differentiation. In

addition, the SII was determined to be an independent factor for length of stay in hospital.

Conclusions

The results of this study demonstrated that the SII could be predictive in the determination of disease severity in AP patients, and was one of the independent factors in the duration of hospitalization of patients.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

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Conflict of interest

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